THE AMERICAN JOURNAL
OF
OPHTHALMOLOGY

VOL. XXX. MARCH, 1913. No. 3

ORIGINAL ARTICLES.

ON TUBERCULOSIS OF THE EYELID.*

By Harry Friedenwald, M.D.,
BALTIMORE, MD.

One of the distinct advances in ophthalmology that has been made in recent years has been the recognition of the importance which tuberculosis plays in disease of the different parts of the visual organ. Tuberculosis of the eyelids, however, has received but slight consideration in English and American ophthalmic literature. Parsons in 1904 (Pathology of the Eye, Vol. I. Part 1, p. 6) discusses the subject in a few lines under the head of "Lupus," though he gives an excellent account of tuberculosis of the conjunctiva. Recent American text-books follow the same lines. As a result we do not consider tuberculosis as an ætiologic factor when small areas of infiltration or ulceration of the eyelids are met with unless there is a clearly marked appearance of lupus.

The German and French handbooks make a clinical distinction between the various forms of lid tuberculosis. Terson in the Encyclopédie Francaise d'Ophtalmologie (1096, Vol. 5, p. 422) distinguishes "two chief varieties, tuberculous ulcerations, granulation-vegetation and abscess on the one hand,—and lupus on the other." Under the head of tuberculosis proper lesions are described which are rarely primary and usually spread from neighboring regions. It is necessary to differentiate tuberculous lesions from abscess, chalazion, syphilis and actinomycosis.

*Read before the Ophthalmological Section of the Medical and Chirurgical Faculty, December 18, 1912, at which time Case 2 was demonstrated.

A fuller account is found in the second edition of Graefe-Saemisch's Handbuch, Vol. 5, pt. 2, p. 94, from the pen of Michel. He adopts on the whole the division of skin tuberculosis laid down by Mracek.

1. Lupus (presenting yellowish gray or brownish red nodules from the size of a pin head to a hempseed).

2. Scrophuloderma (in which the lesion usually begins in the lymph glands or lymph vessels, i.e., "cold abscess").

3. True tuberculous ulcer (the miliary tuberculosis of the skin).

4. Tuberculous warts,* and

5. Tuberculous fistula.

Infection may be metastatic, but is commonly direct; may attack those otherwise free from tuberculosis or may be self-infected. He finds that tuberculous ulceration is rarely primary and describes it as follows: "There are at first slightly raised tubercles or milium-like formations, which rapidly break down and become ulcerative, at which time they are easily mistaken for chalazion. The floor of the superficial ulcer is coated with soft yellowish-red granulations, the margins are sharply cut, jagged, very congested and but slightly infiltrated. In the immediate neighborhood fresh tubercles may develop. . . . . The margin of the lid is the part most liable to the affection, especially the median portion of the lower lid. The destructive process along the margin of the lid may become very extensive. The disease may spread to the conjunctiva but it rarely spreads far over the surface of the lid. . . . It may spread to the opposite part of the other lid. The preauricular glands become somewhat enlarged. . . ."

Case 1.—Mr. J.E.N.S., age about 60, from Virginia, consulted me August 6, 1910, because of a circumscribed inflammation involving the inner half of the right lower lid, from which he had suffered for some time. I found the margin of the lid thickened and inflamed and somewhat ulcerated, and the skin and conjunctival surfaces rough, looking much like an inflamed chalazion. (See Fig. 1.) Yellow oxide of mercury ointment was ordered. I saw him at intervals of several weeks and found that it did not improve under this treatment. It assumed more and more the appearance of an incipient epithelioma and it was because of this suspicion that I advised the use of the X-ray treatment at

*Michel places this class under the head of Lupus and introduces "Solitary tubercle with calcareous degeneration."

the hands of Dr. Cotton. Preliminary to this, I removed a small mass from the inner surface of the lid and received the following report from Dr. Standish McCleary:

"This tissue mass, a little larger than a pin head, is covered with epithelium for a short distance at one margin. The rest of the surface shows ulcer with a granulating base. In the tissue are seen at least half a dozen tubercles. The intervening tissue consists of a fibrous stroma rich in cells. There is evidence of fibroid change in the tubercles and typical giant cells are seen there. In this specimen no attempt was made to demonstrate the presence of tubercle bacilli by special staining."

The patient showed no evidence of tuberculosis elsewhere and was otherwise quite well. There was no enlargement of the preauricular glands. The X-ray treatment was given in all about six times with varying intervals, medium vacuum tubes be-

FIG. 1.—J. E. N. S., August 6, 1910.

ing used; the exposures lasted 8 to 10 minutes and the distance from the tube was 10 to 12 inches. The applications were discontinued when marked reaction occurred.

During the course of this treatment a number of pustules appeared which were opened and touched with formaldehyde. The improvement was slow but ended in complete recovery.

Case 2.—Miss R. O., aged 13, was brought to my office August 13, 1910, while I was on a summer vacation and was seen by my assistant. The child had suffered from severe ophthalmia neonatorum. When she was a child of 5 or 6 there had been tearing of the left eye and the canaliculus had been slit completely. The right eye had been inflamed for a couple of months; the left eye for four days. There was marked photophobia and lacrimation, with clouding of the cornea; but there was no staining after instilling fluorescine solution. The diagnosis was interstitial keratitis. There was no evidence of inherited

lues. The mother had borne eight children of whom the patient was the third oldest. The fifth child had died as an infant and the seventh birth was premature, the infant dying. The teeth are well formed. There is no history of tuberculosis in the family. Under iodide treatment and under local treatment of atropia and later dionin, the cornea cleared up and the patient's vision improved so that she was discharged from treatment as cured in the spring of 1911. In December, 1911, she returned and showed a circumscribed inflamed and swollen area around the inner canthus of the left eye. (See Fig. 2.) On inquiry it was learned that this had been present for three months. The inflamed area embraced the canaliculus which had been slit years before. The swollen area was smooth, showed no granulations or other irregularities, nor was there any evidence of ulceration. The line of demarcation was not sharp. On the con-

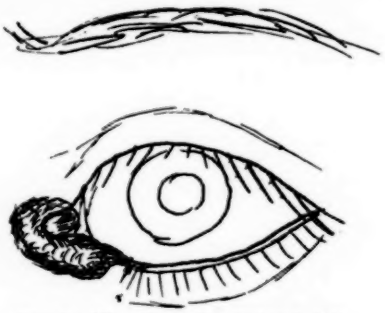


FIG. 2.—Miss R. O., December 3, 1911.

junctival surface the redness and swelling extended a slight distance, but here, too, there was no evidence of ulceration. Excepting for its peculiar location and its being limited to a small part of the lid, it had all the appearance of a chronic blepharitis with marked thickening. Iodide of potash was given; the yellow oxide of mercury and ammoniated mercury ointment were used without showing any influence on the disease. In October, 1912, a piece of tissue from the skin surface was removed and the cut surface sutured. The excised piece was examined by Dr. McCleary. At the same time Dr. Cotton was requested to apply the X-ray treatment. He made similar applications as in Case 1, but used an aluminium screen for protection. After about twelve treatments, made twice weekly, marked reaction set in and the applications were discontinued. In the meantime the swelling and infiltration disappeared, and at the present time the area formerly affected shows only slight

congestion.* The pathologic report of Dr. McCleary is as follows:

"There is no ulceration present in this growth, the surface being covered with sound epithelium. In the tissue numerous tubercles are to be seen, some of which are typical throughout, while in others lymphoid cells predominate over the epitheloid elements. Giant cells are frequent. Around some of the tubercles a fibrous wall has been built up. There is little tendency to caseation. Efforts to demonstrate tubercle bacilli were ineffectual. In as much as bacilli are in so scant numbers in these tissues, this is not surprising. Diagnosis: Tuberculosis of skin."

*This has disappeared completely.

A CONVENIENT FORM OF THE HAITZ STEREOSCOPIC CHART FOR THE INVESTIGATION OF CENTRAL SCOTOMA.

By J. W. Charles, M.D.,
St. Louis, Mo.

In the October, 1904, number of the *Klinische Monatsblætter fuer Augenheilkunde,* Haitz described a new method of charting the limits of central scotomata by means of the stereoscope. This method is based upon the fixing ability of the other eye and the natural inclination to binocular vision, its great advantage

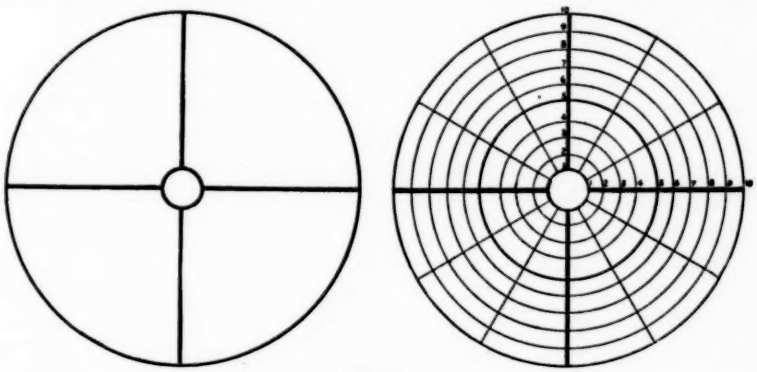


Fig. 1.

being that if the fusion sense of the patient is at all developed, the examined eye will fix much more accurately than under the old method of examination with the other eye covered. As Haitz expressed it, "This is especially true when there is a very small central scotoma. Every experienced ophthalmologist can recall

the difficulties in getting the patient to keep his eye constantly looking at the center of the arc in ordinary perimetry. This method of course requires central vision and fixing ability in the good eye. One would not think of using this method in a case of double macular disturbance."

Wishing to use a chart which would more nearly correspond to our record- and perimetry-cards, and which would not require a waste of time in copying from his charts to a more permanent form, I have drawn the following to be used with

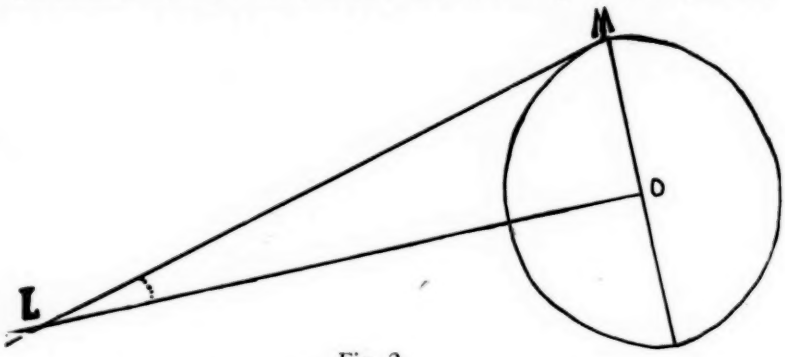


Fig. 2.

In the figure, MOM represents any of the degree-circles on the stereoscopic chart (with MO as radius) placed in the focal plane of the lens situated at L.

LO=focal distance of the lens. $\frac{MO}{LO}$=tan MLO.

any cheap American stereoscope, as advised by Haitz, using his formula $\frac{MO}{LO}$=tan MLO. If the value of the lens is 5D, its focal distance LO=20cm. Therefore MO (the radius of the degree-circles on the chart)=20×tan MLO. For the values of tan MLO from 1° up to 10° we consult the table of natural tangents and find that the following values result, the value of MO depending entirely upon the focal distance of the lens LO: Since

tan 1°=.01746 then for 5D(20cm), MO=(×20)=.03492 cm
tan 2°=.03492, " " " " =.06984 cm
tan 3°=.05241, " " " " =.10482 cm
tan 4°=.06993, " " " " =.13986 cm
tan 5°=.08749, " " " " =.17498 cm
tan 6°=.10510, " " " " =.21020 cm
tan 7°=.12278, " " " " =.24556 cm
tan 8°=.14058, " " " " =.28116 cm
tan 9°=.15838, " " " " =.31646 cm
tan10°=.17633, " " " " =.35266 cm

With MO as radius draw the circles as above with the compasses.

HYALITIS CAUSED BY (*a*) PUS-ABSORPTION AND (*b*) INTESTINAL AUTO-INTOXICATION.

By J. W. Charles, M.D.,
ST. LOUIS, MO.

In the American Journal of Ophthalmology, May, 1912, McCubbin and Gundlach reported "A Case of Optic Papillœdema and Paresis of the Third Nerve Caused by Inflammation of the Ethmoidal Sinuses" on the left side with recovery after complete emptying of the sinuses. The following is a brief history of the case before the operation:

The patient had come to me October, 1910, with severe frontal headaches, worse mornings, which had troubled her for 7 years. Her condition then was O.D. or O.S. Hm. 1, vision=19/12; pupil normal in every reaction, fundus normal except that the veins were larger than usual in contrast with the arteries; disc margins perfectly defined. On account of ethmoidal tenderness and slight fulness, and the fact that she had used glasses without relief, she was referred to Dr. Greenfield Sluder, who examined her for several days without discovering any sinus inflammation. She disappeared before he was satisfied with the exhaustiveness of his examination, and in April, 1911, six months afterward, Drs. McCubbin and Gundelach found her suffering with left internal ophthalmoplegia and both disc margins slightly blurred. Vision in the right eye was 20/24, with the left 20/48. Temp. ranged from 101 to 102.8.

There was improvement after an operation opening the anterior and posterior cells, but the patient returned in a few weeks with severe headaches; vision as before the operation, although the disc-margin of the right eye was more easily defined than before. Ocular movements were perfect in all directions except convergence of O.S. Pupil of that eye was moderately dilated. Two days later, while under daily observation, the vision of the left eye sank to 20/240 to 20/150; the pupil was widely dilated, but a few hours later was normal and reacted to ordinary daylight. Color fields were normal, as well as form-fields, with O.D.; but the left eye failed to differentiate colors. Temp. was 99.6.

On May 10th, the anterior and posterior ethmoidal cells with the frontal and sphenoidal sinuses were widely opened, and on May 11th the headache was less and the pupil somewhat smaller.

Temp.=100. In six days no headache for the first time in years. Temperature remained above normal on June 17th, the afternoon temperature of 100 persisting. Fundus and vision normal.

On July 5th, the patient had an abscess of the cheek following picking a pimple. This was drained and dressed.

July 8th: Had two days before a severe headache of half an hour duration relieved by the evacuation of "several teaspoonfuls" of pus from the left nostril. O.D. with+0.25 cyl. ax. vertl. V=20/15+. O.S. with+0.25 cyl. ax. 115 V=20/15+.

July 10th: Was sent to the Washington University Hospital with appendicitis by Dr. P. G. Hurford. She was discharged August 9th without an operation. Temp.=101.3. Nose in good condition on August 10th.

September 22nd: The patient reported improvement in every respect and that she had gained in weight until September 16th, when she noticed a swelling in the left cheek. The finger in the mouth shows a distinct swelling in the canine fossa. Teeth in good condition. Transillumination show antra clear. Nose in good condition.

February 23rd, 1912: The patient reports to Dr. Gundelach and myself that she has been comparatively free from headaches all winter. Vision=19/15 R. or L. Eyes have ached in near work. After several examinations ℞ +0.37 cyl. ax. vertl. R. and L. and return for full examination.

April 10th: The patient did not return until to-day. Used atropia gr. 1 to ℥ii.

April 11th: After atropia 1/120 R. and L. O.D HO.62 V=19/15; O.S. HO.62 V=19/15. ℞ +0.75 Sph. R. and L.

October 16th: Comes with the single complaint of eyes discharging a great deal. Rather vague statements concerning headaches. Without a mydriatic O.D. Em V=19/15, showing slight spasm; O.S. Em V=19/12. Pupils normal. Ophthalmoscope shows O.D., punctations on Descemet's membrane slight, but distinct hyalitis and the nasal margin of disc rather blurred. No lesions visible in chorioid or retina. Veins rather large.

October 21st: The disc-margin of O.D. is distinctly more blurred than that of O.S. Hyalitis is more marked and the punctations on the membrane of Descemet are more numerous. V=as 16th. Pupils are normal in every respect. The patient has intense headaches—both sides. The patient states that her temperature is often 99° to 100°.

The patient was then referred to Dr. Albert E. Taussig for

a thorough investigation, in hospital environment, of the patient's general condition. She was admitted to the Hospital of the Medical Department of Washington University October 25th, 1912, where she was kept in bed under observation until November 22nd. During this time her temperature was almost constantly above normal, rising in the afternoon to 101° to 103°. Physical examination gave normal findings. Urine and fæces normal. Wassermann negative and blood culture negative. Tuberculin subcutaneously was not feasible on account of heightened temperature. (In August, 1911, von Pirquet had been "dubious".) The blood count yielded a leucocytosis, the white cells ranging from 10,580 to 12,640. Dr. Taussig reports that this leucocytosis "was strictly one affecting the polyneuclear neutrophiles, the lymphocytes and other cells being always normal in proportion."

On Oct. 23rd, Dr. R. D. Carman took X-ray plates which gave "a possible sinusitis of the left antrum. It is much smaller than the right, the septum being markedly deflected to the right. Sella turcica is normal."

On November 3rd, Dr. Sluder found a suppurating sphenoidal sinus on the left side, but did not believe that it explained the patient's fever. He operated November 12th, thoroughly cleaning out the source of pus.

In spite of the perfect picture of pus-absorption, with its leucocytosis, the patient continued to have fever and to feel miserable.

On November 22nd, she came to the office and her vision was found to be 19/24 R. or L. Emmetropia without cycloplegia. She complains of constant headaches with "striking pains". She says that her temperature was 103 last night. Goes home for recuperation.

December 6th: Temperature remains about 100. O.D. V= 19/19; O.S. V=19/15.

January 21st, 1913: O.D. Em V=19/19+; O.S. V=19/19+. Pupils are normal in every respect. The ophthalmoscope gives slight improvement in the appearance of media and disc-margin. The weight of the patient has increased "ten pounds", and fever has disappeared. ℞ Bichloride gr. 1/12 t.i.d. The patient did not return this afternoon for an examination of her fields as directed.

January 29th: The patient reports complete comfort for the last eight days. Her appetite is improving and she is taking care of two young children.

T. A. H., 41 years old, came February 14th, 1911, with vision failing in right eye for some weeks, the history being rather indefinite. Ophthalmometer gave AS 0.5 M vertl. R. and L. Ophthalmoscope gave O.D. vitreous very muddy; O.S. normal. Trial gave O.D. AH 0.5 M 165 Hm 0.25 V=19/38; O.S. Ah 0.5 M Horl. Hm 0.5 V=19/12. Gave note to Dr. H. W. Soper advising bichloride if, after an exhaustive examination, there was yielded no contraindication.

March 20th, after five weeks of treatment with bichloride and purin-free diet: O.D. V=19/30+; O.S. V=19/12+.

April 3rd: O.D. V=19/24; O.S. V=19/12.

May 16th: O.D. V=19/24+.

October 2nd: O.D. V=19/24 to 19/15.

December 31st, 1912: There has been no appreciable change in appearance of fundi nor in the vision, since last examined in October. Dr. Soper's notes follow:

January 27, 1913.

T. A. H., male, age 41 years, married. Occupation, stock man with commission company. Came under my observation December 1, 1910. Drinks an occasional glass of whiskey or beer, but no regular habit. Smokes both pipe and cigars four to five times daily; does not inhale. Drinks one cup of coffee and one cup of tea a day. He has never had any serious illness. Denies all venereal diseases. For the past twelve years has had stomach trouble from time to time. Has definite attacks of gaseous distension and sore feeling in the epigastrium, usually followed by diarrhœa; no nausea or vomiting.

One year ago had a definite attack of appendicitis and the appendix was removed after the attack had subsided. His stomach trouble was better after the operation, until two months ago, when he developed a cough and considerable discomfort in the stomach region three or four hours after eating. Salads and acids seemed to increase the discomfort, which was not a pain but a sort of a burning feeling. A glass of hot water or hot milk caused immediate relief. The cough became worse during the last week, accompanied by some fever and considerable expectoration of muco-purulent material.

He is a man of large frame, 5 ft. 10½ in. in height, weighs 170 lbs. (three months ago he weighed 185 lbs.).

Physical examination of the chest disclosed the presence of sibilant and sonorus râles over both sides posteriorly. Pulse 80, regular, good volume, compressible. Blood-pressure 110; tem-

perature 99 degrees. The abdomen somewhat distended. The entire epigastrium tender to pressure. No enlargement of the liver or spleen. Kidneys not palpable. The urinalysis (24 hour quantity 2000 cc.): Sp. Gr. 1018, yellow in color, acid reaction, albumin faint trace neucleo, no serum, no sugar, no bile. Large amount of indican, no casts, no blood or pus; few leucocytes.

Feces: Acid reaction, dark brown in color, 150 g. well formed. Many large clumps of mucus, no visible blood, no occult blood.

Sputum showed many pus cells. No tubercle bacilli.

Stomach contents after a test breakfast, 30 cc. yellowish liquid, chym fairly good, considerable excess of stomach mucous. Free HCl 50, total acidity 70.

A diagnosis of catarrhal bronchitis, gastritis hyperacida, and general auto-intoxication was made.

Patient stated that dust in the stock yards where he worked was very great and he attributed his bronchitis to this. He was put to bed for a week and the bronchitis improved rapidly; the indican disappeared from the urine in four weeks under a strictly purin-free diet. He quit the stock yards and went to work on a farm.

February, 1911: Dr. Charles discovered opaque bodies in the vitreous. Bichloride of mercury in doses of 1/12 to 1/5 gr. three times daily was given and continued to date.

November 10, 1912: Wassermann reaction negative. Dr. R. Buhman. Von Pirquet *weak* positive, "practically negative." Dr. R. Buhman.

Patient now weighs 182 lbs. and is in splendid physical condition.

The weak spot in the report of both these cases is that the one had von Pirquet only done, and that as far back as 1911, and her condition was such that Dr. Taussig did not believe it advisable to give a subcutaneous diagnostic tuberculin test; and the other case had a weak positive reaction to the von Pirquet test which Dr. Buhman asserts was practically negative. These weak points will be strengthened as soon as possible, but the diagnoses seem so certain without this procedure that I feel that absorption of pus in the one case and auto-intoxication in the other were the causative factors.

MEDICAL SOCIETIES

TRANSACTIONS OF THE PHILADELPHIA POLYCLINIC OPHTHALMIC SOCIETY.

January 9, 1913.
Dr. Wendell Reber in the Chair.

EXHIBITION OF CASES.

Dr. William Campbell Posey showed a man from whom he had recently removed a large carcinoma from the orbit with preservation of the globe. Although it had been necessary to enlarge the incision proposed by Krönlein by a horizontal one which extended below the superciliary ridge and parallel to the supraorbital margin to permit the removal of the growth, the deformity was exceedingly slight, the lines of incision being barely visible.

A Tansley-Hunt operation had been performed some months after the Krönlein procedure to relieve the ptosis occasioned by the loss in function in the levator consequent upon the division of the nerve supply. Full details of the clinical history and the pathological report will be given later.

Dr. Reber called attention to the excellent motility of the globe and the splendid position of the eyeball and stated that it was the most perfect result after a Krönlein operation that he had ever seen. One really had to hunt for the scar left by the temporomalar incision.

Dr. Reber exhibited a case of a young colored woman, 18 years of age, with a violent bilateral uveitis anterior, reducing vision to about 1/60 in each eye. She had lived to the flesh with all her might and main; result, a strongly positive Wassermann reaction.

The left eye had shown a very small scleral staphyloma from 2 to 3 mm. in diameter just back of the upper outer limbus but it had subsided promptly on treatment. The right eye then began to develop hypertension with great supraorbital pain and narrowing of the anterior chamber. This became so marked that a paracentitis was done with consequent prompt reduction of the tension, but after a few days a scleral staphyloma began to bulge forward at the lower limbus and in spite of all medical treatment

continued to increase in size until it was larger than the cornea itself. During all this time the tension remained about minus one, and the patient was absolutely free from pain. The patient was seen in consultation with three or four colleagues, all of whom inclined to the diagnosis of gumma and two of them were quite convinced that enucleation would become absolutely necessary. Neo-salvarsan and mercurial inunctions had been used for about two weeks without any benefit. At this juncture the eye was thoroughly cocainized and 3 drops of absolute alcohol (95 per cent.) were introduced directly into the substance of the ectasia by means of a hypodermic needle. Within five days the ectasia was hardly in evidence and within ten days there was nothing to mark the site of the previous extensive staphyloma other than a broad pigmented line along the lower limbus. Dr. Reber speculated upon the possible conditions that existed within the ectasia, rejected the idea of gumma and thought it might have been a cystic condition.

Dr. Posey in discussing the question also rejected the idea of gumma, although he saw objections in the way of assuming that the condition was a cystic one.

The Use of Bifocal Glasses in the Orthoptic Treatment of Strabismus in Children.—Dr. Wendell Reber.

Dr. Reber discussed the various theories for the genesis of convergent strabismus in little children, saying that he was convinced that the usual clinical picture was that of a weak or even entirely absent fusion sense complicated by a more or less unequal refractive error. With a weak fusion sense the accommodative apparatus of the eye easily falls into vicious habits. The correction of these vicious states of the accommodative apparatus have been made up to the present time, by the use of practically full correction and the continuance of atropia in one or both eyes for one to three months after the glasses were put on. Two years ago he had come to the conclusion that it could do no harm to carry this idea to its logical sequence. He therefore not only ordered a full correction for infinity, but to this added plus 2 to 3 D. to be made up in an invisible bifocal glass, thus setting aside practically all accommodative effort in the eyes of these little subjects, whose accommodation is so abnormally spastic. The combination of bifocal glasses and atropia in both eyes for two to three months is the most complete orthoptic method for the abolition of accommodative spasm, with its associated over-convergence.

The first of such glasses ordered were made up in lenticular bifocals, but they excited so much comment when worn by a 4 year old child and subjected the parents to such unceasing questions from every interested person that the glasses were made up in an invisible bifocal. Bifocal glasses have been tried in this way in 8 cases of seemingly pure functional strabismus. That is to say, no congenital features present in any of the cases.

Dr. Reber has been much impressed by the excellent results achieved in these 8 cases and thinks they are superior to those secured by the methods at present in vogue. One objection is that the little patients at once try to dodge the bifocal and for the first week bob their heads about a good bit, but they soon learn to adjust themselves and the bifocal's good effect upon the deviation seems to follow soon after. He feels that the method is well worth the trial.

DISCUSSION.

Dr. Posey expressed his appreciation of the value of Dr. Reber's suggestion, and said that as Dr. Reber claimed, the removal of the accommodative effort by the superposition of additional spherical strength in the bifocal slips, was quite in line with the teachings of Donders and should tend to establish as far as was possible the normal relationship between accommodation and convergence which was necessary for straight visual axes. The only objection he had to offer was the difficulty there might be in adjusting the slips to the squinting eyes. Dr. Posey was averse to glassing children younger than three years of age and decried the practice of some of tieing the spectacles on the heads of infants with tapes, upon account of the danger of the pressure exerted by the glasses upon the root of the nose, altering the formation of the lines of the face and interfering with the proper development of the orbit. He insisted upon the early differentiation of cases of congenital squint from those under discussion. He doubted if in cases of concomitant squint, vision sank as rapidly in the squinting eye as Worth has averred.

Dr. Zentmayer said that believing as he does in the correctness of the theory of Donders as to the cause of esotropia, he considered Dr. Reber's method rational, but not generally practicable in young children. Exactly the same thing has been recommended for esophoria, that is, to diminish the accommodative effort at near-work by the addition to the distance glass of a plus 2 or 3 D. lens to be worn for near-work.

Dr. Pfeiffer said he had little confidence in Worth's method. Experience had shown him that it required very intelligent parents to permit following out similar plans to Dr. Reber's on children. He thought that with a lower segment, no matter how it was shaped, there attended an effort on the child's part to look around the edge to escape the more powerful glass.

Dr. Reber, in closing: There is no question, as I pointed out in my opening remarks, but that these little patients have difficulty the first week or ten days in getting used to the bifocal glass. In no one of the cases has the discomfort extended beyond this time. The influence of the fusion faculty on the genesis of strabismus in little children cannot be lightly regarded. It is perfectly true, as Dr. Zentmayer says, that the use of a plus 2 to 3 over the infinity correction has been in vogue for some years. In the first edition of Hansell and Reber on the "Muscular Anomalies of the Eye," (1896) this method of the treatment of esophoria was gone into at some length, and during these 17 years we have resorted to the method in a goodly number of cases with gratifying results. Indeed it was the success of these cases which led us to apply the above mentioned method in strabismus of little children. If the object of the orthoptic treatment of strabismus is to break up the vicious accommodative habits into which the visual apparatus has fallen we believe this will prove to be a contribution to the orthoptic treatment of squint.

W. WALTER WATSON, M.D.
Secretary.

[*Continued in April number.*]

CORRECTION.

Dr. W. Franklin Coleman is not located at Toronto, as might appear from the title of his book reviewed in the February number of this Journal, page 63. He resides at Chicago.

THE ROYAL SOCIETY OF MEDICINE.

SECTION OF OPHTHALMOLOGY.

The first clinical meeting of the Section was held on Wednesday, 6th November, under the Presidency of Sir Anderson Critchett, C.V.O.

Mr. Herbert Fisher showed a case of subhyaloid hæmorrhage, with drawing. He urged the abandonment of the term "subhyaloid hæmorrhage," as the hæmorrhage was intra-retinal; he suggested the words "semilunar retinal hæmorrhages." The President agreed with the suggestion.

Mr. A. W. Ormond showed a case of pemphigus of the conjunctiva, followed by essential shrinking. The patient was 24 years of age, and as his general condition as well as his sight were now so bad, he pleaded for something to be done. Mr. Ormond proposed to clear away the conjunctiva as much as possible, and substitute mucous membrane from elsewhere. Mr. Fisher referred to a case of pemphigus of the conjunctiva, which was later under Mr. Lawford's care, in which a vaccine made from the contents of the patient's own vesicles, was administered for some time, but without marked benefit. Mr. Lawford confirmed the fact that there was no definite improvement after the vaccine treatment. Mr. Bishop Harman described a very severe and extensive case, involving larynx, pharynx, mouth, and both eyes, in which no treatment was of use.

Mr. R. Greeves showed a case of paralysis of the third nerve with periodic spasm of irido-ciliary muscles. He said he could not make out any relationship between the movements of the two eyes. He thought the right pupil was a little unsteady, but it seemed to have nothing to do with the contractions and dilatations of the other eye. Mr. Herbert described a somewhat similar case, and suggested an explanation, namely, that a portion of the nucleus of the third nerve was non-existent and the other portion of it was weak, so that it was able to overcome the innervation of the higher centres only after an interval of rest.

Mr. Herbert Parsons showed a case of Mooren's ulcer, with ulceration of the sclerotic, and Mr. Leslie Paton demonstrated a modification of Herbert's operation for chronic glaucoma, in which his object had been, while retaining the simplicity of Herbert's operation, to procure a more permanent result. Mr. Herbert described his own attempts in the same direction, emphasiz-

ing the importance of not reducing the nutrition of the flap too much. The difficulty arose chiefly in subjects who had a very shallow anterior chamber.

Mr. E. Nettleship read notes of a case in which a sarcoma of the choroid was seen as a small spot of disease, but its true nature not recognized, about 20 years before the diagnosis of tumor was made, and 25 years before the removal of the eye. The case showed the importance of watching over long periods, when possible, the behavior of certain solitary spots or patches of dusky discoloration that are occasionally seen in the choroid during ophthalmoscopic examination, some of them probably being the beginnings of malignant growth, although others were, no doubt, congenital and stationary. Such solitary, non-inflammatory patches might sometimes be the counterparts of the minute sarcomatous growths, of which nine or ten examples have been accidentally discovered after death and published during the last few years.

Mr. Nettleship also read a joint communication by himself and Mr. A. Hugh Thompson on an extensive pedigree of Leber's disease of the optic nerves, which illustrated the occurrence of the malady in females, recovery in some cases in both sexes, descent to all the children of one of the affected women, diabetes with blindness of unknown nature in one member, high infantile mortality in the very large family of one affected man, and absence of influence of the optic nerve disease upon prospect of life.

The President paid a tribute to the labors of Mr. Nettleship in the domain of hereditary disease, and referred to the changes of medical opinion on the subject of heredity. Mr. Hugh Thompson supplemented the paper in respect to one patient, who was a heavy smoker, and suggested that in cases of tardy recovery from tobacco amblyopia enquiry should be made as to any connection with Leber's disease.

Mr. A. W. Ormond read a paper on a case of retino-choroiditis juxtapapillaris. The patient was a man, aged 20, who found, on awaking, that he could not see very well with his right eye. He had a little pain in the eye a week previously. On examination there was found keratitis punctata, and a patch of acute choroiditis touching the upper margin of the optic disc, and spreading upwards. Œdema of the retina spread over and beyond the patch. Vessels which passed over the inflamed area were partly obscured, and the arteries diminished in size; there

was also some haze in the vitreous. Von Pirquet's reaction was positive. The inflammation gradually subsided, and the patient now had full visual acuity, but a large sector of his field of vision, stretching from the blind spot to the extreme periphery, was entirely absent, and he had no perception of light in this area. The defective area in the field of vision was clearly due to the obliteration of a branch of the central retinal artery by the pressure of the inflammatory swelling. Under the title retino-choroiditis juxtapapillaris Professor Jensen, of Copenhagen, published four similar cases in Graefe's Archives, in 1909.

ROYAL SOCIETY OF MEDICINE.

SECTION OF OPHTHALMOLOGY.

December 4, 1912.

Sir Anderson Critchett, Bt., C.V.O., President of the Section, in the Chair.

Discussion on the Physiology of the Intra-Ocular Pressure.—Opened by Leonard Hill, M.B., F.R.S., and E. H. Starling, M.D., F.R.S.

Dr. Leonard Hill, F.R.S.: Some researches which Dr. Flack and I have been carrying out on the relation of the circulation to secretion led us to investigate the eye, and the results of our investigation have been published recently in the *Proceedings of the Royal Society;* these we propose to bring before you to-night. The first task we set before us was to try and measure the intra-ocular pressure. Mr. Thomas Henderson and I made preliminary experiments. We were not satisfied with using the ordinary hypodermic needle, for we could not avoid leakage in the track of such a needle. That led to my devising a special needle, a sketch of which I show you on the screen. Dr. Flack and I have employed this in our measurements on the intra-ocular pressure. It is made of two tubes. The inner tube can be rotated by means of a handle, and there is an eyehole in it which corresponds, in a certain position of rotation, to an eye-hole in the outer tube. When the two holes are opposite the aqueous humor can pass into the inner tube. If we rotate the handle right round, the eyehole of the inner tube is no longer in

correspondence with the eyehole in the outer needle, and the needle is closed. As we put it in closed, no aqueous can escape. We have a conical shaped needle, and it goes in as a cone, and that prevents leakage, and when it is in, we rotate the needle, until the eyeholes correspond. The aqueous pressure is measured by the compensatory method which I employed for measuring the pressure in the brain. The inner tube of the needle is connected with a length of glass tubing of fine bore which in its turn is connected with a pressure bottle and a mercury or a water manometer. An air bubble is introduced into the glass tube, and the position of it marked when the pressure is at zero, that is to say, at atmospheric pressure. The zero position is obtained when the eyehole is open in the needle, and the pressure bottle is lowered to such a point that the water within it is at the same level as the eyehole, and ceases to flow out. Having marked the position of the air bubble on the glass tube, we push the closed needle into the aqueous, and transfix the anterior chamber with it so that it is steady. We support the needle so that it exerts the least possible tension on the eyeball, and then rotate the handle so as to open the needle. The air bubble is pushed out, and the moment it is pushed so, we bring it back to the zero mark, by raising the pressure bottle, and thus compensate the pressure of the aqueous without letting any of this fluid escape. Then we read the pressure. When inserting the needle we try to get as near the edge of the cornea as possible, and push it right through. The hole lies in the aqueous, and we make sure it is free in the aqueous, and we can see it in there. It is very important to support the needle, because any traction on the eyeball will raise the pressure of the aqueous, and that is a great source of error. Mr. Thomson Henderson has suggested another source of error—namely, that the introduction of such a needle into the eyeball may cause some reaction in the eye; a temporary obstruction of the circulation occurs during the introduction of the needle, and a subsequent dilatation of blood-vessels probably results, which raises the pressure to a greater level than the normal. Such a reaction would soon subside. We have controlled the reading of the needle in this way. We pushed into the same eye or into the opposite eye a hypodermic needle, a very small one, connected with a gauge which I use for measuring blood-pressure. The central readings taken by means of this gauge show that the aqueous pressure varies within fairly wide limits, depending upon the blood-pressure. The higher

the arterial pressure, the higher is the aqueous pressure. We have got readings as low as 16 mm. Hg. in one cat under chloroform and with a low arterial pressure, going up under ether narcosis, when the animals were in good condition, as high as 62 mm. Hg. Variations of the kind, agreeing between arterial and aqueous pressure, left no doubt in our minds on the point. In comparing the readings with the hypodermic needle with those with our needle, done by the compensation method, we got readings which seemed to agree. So we could not find much source of error.

So much for the aqueous pressure. We do not very much mind what the aqueous pressure is, as it is not of very great importance except that it is positive. It is very important that it should be positive, because that keeps the eyeball as a perfect optical instrument. The eyeball is distended, in our opinion, by a secretory pressure, and that keeps it as a perfect optical instrument, perfect in its shape, equally distended all over. And to preserve that, positive pressure is required, and it does not seem to matter very much whether that positive pressure varies from 10 mm. to 50 mm. Hg., for the outer coat of the eyeball is firm enough to resist any distension, and the eyeball remains the same shape and size under very different strains. When a man is making an effort he may have an arterial pressure of 150 to 200 mm. Hg. The pressure of his aqueous may then be 50 to 70 mm. Hg. When he is at rest his arterial pressure will be about 100 to 120 mm. Hg. and his aqueous 20 to 30 mm. Hg. We accept the common theory that the aqueous is secreted by the choroidal fringes, and we maintain that the aqueous is practically at the same pressure as the capillary venous pressure in the choroidal fringes. There is nothing between these fringes and the aqueous but layers of protoplasmic cells containing some 80 per cent. of water. The capillaries are enclosed by a transparent, excessively thin membrane, the capillary wall also is composed of wet protoplasm. We do not know how much water this protoplasm contains. Connective tissue and membranæ propriæ limit the expansion of the cell protoplasm, but we find no evidence of structure which can hold the capillaries open when pressed on by the aqueous from outside. Capillaries elsewhere in the body are collapsed and emptied with the greatest ease by pressure applied outside—e.g., in the skin and brain. We maintain that the pressure of vitreous and aqueous, which has been proved to be always the same, is equal to the pressure in the

veins at the place of exit from the eyeball. If it was greater, the veins would be shut up. There is nothing in the wall of the veins which can support pressure from the outside. So we maintain that the pressure in all the various venous exits must be the same as the intra-ocular pressure, and the pressure therein and in the capillaries is practically identical. The least (immeasurable) difference aided by the pulse (transmitted through the contents of the eyeball) will suffice to maintain the flow from capillaries to veins. Muscular contraction of intrinsic and extrinsic muscles of eyeball also maintains capillary-venous flow as elsewhere in the body. One of the experimental proofs which we bring forward with regard to this point is this: If we open one of the vortex veins in the eye of the cat, allow the blood to escape from there, and force Ringer's solution to run into the aqueous, by raising the pressure bottle, the outflow from that vein stops when the aqueous pressure is raised to the arterial pressure. This shows that the two pressures go up together; the aqueous pressure and the capillary pressure rise together, and when you reach the arterial pressure the outflow stops. It is the same as putting an armlet and sphygmomanometer on the arm, and gradually raising the pressure of the armlet to nearly the arterial pressure; the veins will get fuller and fuller until the pressure in the veins gets to the same as the arterial pressure, or almost the same. So we put in the fluid and raise the pressure in the eyeball: we finally stop the venous outflow, and the pressure required to stop the flow is the full arterial pressure. That is confirmed by observations made by a different method. The method is this: Looking at the back of the eyeball, and watching the flow in the retinal vessels, another observer, v. Schulten, found very much the same thing: that the flow ceased, as viewed from the ophthalmoscope, when the pressure reached the arterial pressure. This shows, to our minds, that the two pressures go together, and that if you raise one you raise the other.

I now show you two photographs of cat's eye, taken after an experiment made to-day. Afterwards I will show you in the epidiascope the actual eyes. The experiment we did is a very easy one to perform, and very striking and interesting. We simply punctured the cornea of one side, and allowed the aqueous to escape. On then gently pressing on the belly of the cat, the iris on this side immediately burst into hæmorrhage; all the capillaries allowed blood to escape. That comes off every time. The explanation is that the normal aqueous pressure is exactly

counterbalancing the capillary pressure. If you allow aqueous to escape and then press the belly to raise the arterial pressure, the capillaries at once burst, and hæmorrhage takes place into the anterior chamber. That shows that the two pressures are one and the same, the one supporting the other, and explains why there is hæmorrhage in operations and injuries to the eyeball. I do not know whether the explanation is new to you as ophthalmic surgeons; it may be that all of you have already recognized that that is the explanation. It is perfectly clear that the aqueous no longer supports these blood-vessels, and therefore when the blood-pressure is raised they burst, and hæmorrhage takes place.

The aqueous has a distinct chemical nature, and its osmotic pressure is said to be higher than that of the serum. And we know that if fluorescin is put in intravenously, as Ehrlich did, it is secreted, and appears in the anterior chamber, and can be demonstrated there. That shows that some kind of circulation is going on in the aqueous. But to-day I saw a record of some observations, which showed that if fluorescin were put in so that there was one part in 20,000 circulating in the blood there was found one part in 400 in the urine, and one part in a million in the aqueous fluid. Therefore you see it does not get concentrated in the aqueous and the method does not show that the aqueous is being rapidly secreted and absorbed. The observations of Ehrlich are very beautiful, showing as they do the secretion of the aqueous, and the very important fact, which is known to all workers on the eyeball, that if the anterior chamber is pierced, the subsequent secretion is quite different and is far more intensely tinged with fluorescin; it is one which contains blood corpuscles, is rich in albumin, and like serum. It is a leakage secretion. So we maintain that the secretion, or absorption of the aqueous, cannot be studied in any way by opening the eyeball, or allowing the aqueous to escape, because the conditions are entriely altered. We must have a closed eyeball, with the aqueous pressure balancing the capillary as it naturally does.

I pass now to the consideration of the fluid of the eyeball in relation to accommodation. I want to deal with the transference of fluid from in front of the lens to the side of the lens, which we think is a most important point in accommodation. In whatever way it may be brought about there must be this transference of fluid, and the mechanism of accommodation is thereby coordinated in the most delicate way, because the transference of

the least quantity of fluid can bring about instantly, in a most beautiful way, the changes in the shape of the lens.

It is always said that the lens capsule and the suspensory ligament are in the eye, exerting elastic pressure on the lens, and causing it to assume its less convex shape. What is the cause of that shape? We maintain it is the intra-ocular pressure; it is the pressure in the aqueous and the vitreous maintained by the secretory action of the choroidal fringes. The contraction is equal in all directions in the eyeball, which pulls on the suspensory ligament and keeps this lens in its natural shape. And if you cut out the lens from the body, it increases its curvature, and why? Because it is taken from the influence of the intra-ocular pressure, and yet many people say the lens alters its shape by virtue of its elasticity. But the lens, when cut open, does not seem to have any elasticity at all; it is a soft, pudding-like body with no inherent elasticity. We say it is kept in its flattened shape by intra-ocular pressure. It alters its shape afterwards, when it is removed from that pressure. The eyeball is equally distended by the intraocular pressure in all directions, and it does not seem possible to us that the choroid should be pulled forward at all. What we think happens in accommodation is that the muscle contracts, and when a muscle contracts it does not diminish in size, but it occupies the same volume as uncontracted muscle. And when the ciliary muscle contracts it moves inwards, exactly as described by Henderson, and it may also move forwards, as Starling describes in his text-book. This allows the fluid to pass from in front of the lens either into the grooves of the ciliary body, or into the spaces of the cribriform ligament, and into the supra-choroidal space. We cannot, dogmatically, say where this fluid goes, because we have not made a special study of the structures of the eyeball from this point of view. But we maintain that the fluid must pass from in front of the lens to the side of the lens. It must do so to satisfy the physical conditions; there is no getting away from it, and Helmholtz recognized that in supposing that the angle of the anterior chamber was deepened during accommodation. Thomson Henderson says that anatomical study will not allow of Helmholtz's idea.

Professor E. H. STARLING, F.R.S.: Although I am in full agreement with most of the facts brought forward by Dr. Leonard Hill, I do not agree with him so closely as to the interpretation to be placed on these experimental results. From the practical point of view it is of more importance to know what the factors

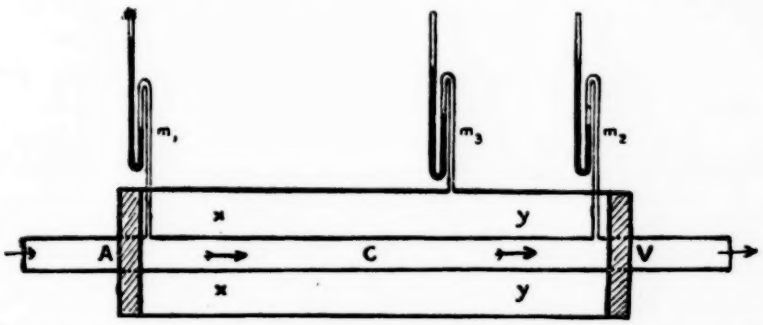
are which determine the formation and absorption of the intra-ocular fluid than to arrive at an agreement as to the theory of its production—whether, for instance, it is due to some mystical process of secretion, some intra-cellular changes which at present we cannot understand, or whether the mechanical processes of filtration are sufficient to account for its production.

Some years ago I took advantage of Mr. Erskine Henderson's assistance to investigate the factors which determine the production and absorption of this fluid. The seat of its production we need hardly discuss, since it is generally agreed that the chief seat of production is the ciliary processes and the chief seat of absorption the anterior angle of the eyeball and the root of the iris. Covering the ciliary processes there is a well-marked epithelium with columnar or cubical cells. These cells are as well formed as those of the salivary glands, or of the kidney tubules, and might therefore easily be assumed to be endowed with secretory powers. Thus *a priori* there is no reason to question the possibility of the intra-ocular fluid being regarded as a secretion.

On the other hand, this epithelium is derived from the optic cup, and is, therefore, a nervous epithelium which is no longer functional. We might consider that it had changed its function and had become a secretory tissue, or that its cells serve simply to support the capillaries and modify the filtering membrane through which the transudation of fluid into the eyeball occurs. In investigating the nature of any process we must start with a hypothesis and proceed from the explicable to the unexplained —from what we can understand to what we want to understand. We must, therefore, in the first place consider what conditions must be observed if the intra-ocular fluid is to be regarded as produced by a process of filtration from the blood circulating in the ciliary processes. In this case the energy necessary for its formation would be derived from the blood-pressure and ultimately from the heart, and not from secretory work effected by the cells covering the ciliary processes. The intra-ocular fluid in the eye is at considerable pressure; there must, therefore, be a resistance to its outflow from the eyeball, a resistance which can only be overcome by a certain pressure. But if the fluid in the eyeball is formed from the blood in consequence of the pressure of the blood in the capillaries, the pressure of the intra-ocular fluid must rise and fall with the intra-capillary pressure. Professor Hill has already told you that the intra-ocular pressure varies with the blood-pressure, and this fac

was well brought out in the work that I carried out with Mr. Henderson. In every case where we raised the blood-pressure in the vessels of the eyeball the intra-ocular pressure went up proportionally and remained high so long as the blood-pressure remained high. It is not merely a question of dilating the vessels by a rise of pressure and thereby increasing temporarily the intra-ocular pressure. So long as the blood-pressure is high the intra-ocular pressure remains high. On the other hand, on cutting off the blood supply to the eyeball the intra-ocular pressure rapidly falls and the eyeball becomes flaccid.

With regard to the normal height of the intra-ocular pressure, I find it difficult to understand how Professor Hill obtained the very high figures he has given us, namely, 16 mm. Hg. to 62 mm. Hg. I have certainly obtained intra-ocular pressures approaching 50 mm. Hg., but this was with exceedingly high pressure in the blood-vessels of the eye. Ordinarily, however, the



blood-pressure is much lower, and we concluded that in a normal unæsthetized animal the average pressure would be not more than 25 mm. Hg.; and a similar figure is given by about a dozen authorities cited by Leber. I think most ophthalmologists would regard a pressure above this point as abnormally high. Not only does the intra-ocular pressure rise and fall with the general blood-pressure, but it can be shown to depend on the local blood-pressure. Thus, if the sympathetic nerve of the neck be stimulated a double effect is produced—first a contraction of the unstriped muscles of the orbit with a rise of pressure, and then, as the vessels of the ciliary processes contract so as to diminish the pressure in the capillaries there is a fall of intra-ocular pressure.

Professor Hill has stated that the pressure in the capillaries and veins of the eyeball must be equal to the intra-ocular pressure. I find it difficult to understand his reasoning. The condi-

tion in the eyeball may be roughly made on a model such as is shown in the diagram, where ACV is the thinnest possible rubber tube passing through the axis of a rigid glass tube *xy*. *xy* contains fluid, and fluid is allowed to pass under pressure from A to V. If the pressure in *xy* is greater than the pressure within the rubber tube, this collapses. If fluid is to pass through ACV it must have pressure all the way along which is somewhat greater than the pressure in *xy*. Now ACV represents arterioles, capillaries and veins, and it is evident that the blood-pressure in these vessels must be somewhat higher than the intra-ocular pressure as represented by the pressure in *xy*. The quicker the rate of flow through the vessels the greater must be the difference of pressure between A and C, and between C and V, and the greater the difference also between the pressure in the elastic tube and the pressure in the space surrounding it. This difference may be of any extent until it is sufficient to burst the rubber tube.

From these considerations you will see that it is impossible to have the conditions assumed by Hill—namely, an equality of capillary and venous pressures. So long as the blood flows from C to V, so long must the pressure at C be greater than the pressure at V.

There is another factor which will tend to exaggerate the difference of pressure between capillaries and veins. Since the kinetic energy of the blood in the veins is greater than that of the slowly moving blood in the wider area of the capillaries, the component of the energy moving the fluid represented by the lateral pressure of the walls must be greater in the case of the capillaries than in that of the veins. Thus a large flow of blood from capillaries to veins involves a considerable difference of pressure between these two points. Professor Hill has stated that the capillaries in the ciliary processes are very delicate and would not, therefore, stand any appreciable pressure. This, however, is a pure assumption, and does not take into account the small cross-section of these vessels. Naturally, if these capillary walls surrounded a balloon a foot in diameter it would burst at the slightest excess of pressure in its interior, but a capillary wall 3 or 4 μ in thickness, surrounding a tube with a lumen 7 μ in diameter, might be expected to stand a considerable pressure before it ruptured. Certainly a bicycle tyre in which the thickness of the wall had the same relation to the lumen of the tube would be strong enough for all practical purposes.

There are no experimental facts which exclude the possibility of a considerable difference of pressure existing between the capillaries and the fluids outside them. Such a difference may be shown to exist in the connective tissue of the limbs. Many years ago, after reading a book by Landerer on the tension of the tissue spaces, I spent several months measuring this pressure. In all cases I found that the pressure was extremely small—namely, 2 or 3 cc. of water, whereas the pressure in the capillaries might be 20 or 30 mm. Hg.

We are not justified, therefore, in denying the possibility of a difference of pressure between the blood in the capillaries and the intra-ocular fluid, and we must assume that, if the circulation is to continue, a considerable difference of pressure exists between the blood in the capillaries and the blood in the veins. The venous pressure itself must be higher than the intra-ocular pressure. If, in the model, the pressure in *xy* is raised to the pressure at V, the tube at V will collapse. As soon as the circulation through ACV is thus brought to a standstill the pressure at V will rise to the pressure at A and the circulation will go on again; that is to say, the circulation through ACV will not be brought to a standstill until the pressure in *xy* is just higher than the pressure at A; which, being interpreted, means that in order to stop the blood-flow through the eyeball naturally, the intra-ocular pressure must rise to the pressure of the blood in the arteries entering the eyeball. That does not, however, tell you what is the pressure in the capillaries of the eyeball under normal conditions; it only gives the upper limit of this pressure and does not mean that the intra-ocular pressure under normal circumstances is equal to the capillary pressure.

Assuming that the intra-capillary pressure is the main factor in determining the production of intra-ocular fluid, it is important to know what is the rate at which fluid is turned out. So long as the intra-ocular pressure remains constant the rate of absorption must be equal to the rate of production. In order, therefore, to measure the rate of transudation, we must measure the rate of absorption at the same pressure. This can be determined by putting a hollow needle into the eye, filled with fluid at such a pressure that the intra-ocular pressure is just balanced, so that fluid neither enters nor leaves the eye. The heart is then cut out so that the pressure in the vessels sinks to zero. No more fluid is now being poured out by the ciliary processes, but

the intra-ocular pressure is maintained at the same height as before by its connection through the hollow needle with a reservoir of Ringer's fluid. The Ringer's fluid therefore flows into the eye, and the rate at which it flows gives the rate at which the intra-ocular fluid was being absorbed just before the death of the animal. In a series of observations made by this method, Henderson and I found that, at the normal intra-ocular pressure, the rate of absorption was about 12 c.m. per minute, and this is, therefore, the normal rate of production of intra-ocular fluid by the ciliary processes.

The proofs I have given you that the intra-capillary pressure of the ciliary vessels is the most important factor in the production of intra-ocular fluid, and that the intra-ocular tension depends on the balance between the production as regulated by the blood-pressure and the absorption as regulated by the resistance at the anterior angle of the eye, do not necessarily imply that the intra-ocular fluid is merely a filtration. It might be that the cells were being stimulated by the blood-pressure, so that the secretion increased or diminished as the blood-pressure rose or fell. Before we can assume that the process is one of filtration we must account for the fact that the fluid, though containing practically all the salts of the plasma, is almost entirely free from protein. The slightly increased molecular concentration of the intra-ocular fluid as compared with blood plasma might be ascribed to metabolites produced by the cells. But if we assume that the capillary wall *plus* ciliary epithelium is a filtering membrane which is impermeable to colloids such as the blood proteins, there would have to be a minimum difference of pressure of about 30 mm. Hg. between the blood in the capillaries and the intra-ocular fluid for any filtration to take place at all; otherwise the blood-vessels would absorb the intra-ocular fluid. Thus the filtration theory demands not only some difference of pressure in favor of the capillaries, but that the pressure in the capillaries shall not be less than 30 mm. Hg. above that of the fluid in the eyeball. Unfortunately, we have no method at present of measuring the intra-capillary pressure in the eyeball directly. It is true that there is always a considerable difference of pressure between the blood in the large arteries and the intra-ocular fluid, and the difference may be from 70 to 90 mm. Hg., but it is impossible to say how much of this pressure in the arteries is transmitted to the capillaries of the ciliary processes. It is impossible, therefore, at the present time to furnish the

crucial proof of the filtration hypothesis. But the strict parallelism which exists between the blood-pressure and the intra-ocular pressure and the fact that with a rise of blood-pressure, the intra-ocular pressure increases, as well as the amount of fluid escaping through the anterior angle of the eye, shows us the intra-ocular fluid merely as a result of balanced mechanical processes; and at the present time there is no evidence of any other processes, and we are not justified in assuming that the epithelium covering the ciliary processes acts otherwise than passively in strengthening and modifying the qualities of the filtration membrane.

We have finally studied the conditions which alter the rate at which fluid escapes from the eyeball. Why does one not always find a permanent rise of intra-ocular pressure with a rise of arterial pressure? The association is certainly seen, but it is by no means constant. The explanation is probably found in some experiments by Henderson and myself. If we try the effect of raising the intra-ocular pressure artificially, we find that a little time after raising the pressure to 50 or 60 mm. Hg., filtration becomes more easy, as if additional channels had been opened up or pre-existing channels enlarged. A similar change might occur in the normal eye, and in this way the intra-ocular pressure might be kept at a normal height, although the arterial pressure was permanently raised. We have also tried in the same way the effect of atropine and eserine on the rate of escape of intra-ocular fluid from the eyeball. At normal pressures no difference was found between the eserined eye and the atropinized eye.

CAT, ANAESTHETIZED WITH ETHER. BLOOD-PRESSURE AVERAGE 138 MM. HG., WITH ONLY TRIFLING VARIATIONS THROUGHOUT THE EXPERIMENT.

Intra-ocular pressure in mm. Hg.	Rate of filtration in eserine eye in cubic millimetres per minute	Rate of filtration in atropine eye in cubic millimetres per minute	Rate of filtration in atropine eye post mortem, in cubic millimetres per minute
20	0	0	12
35	11	8	20
50	16	11	25
65	23	14	31

At 20 mm. Hg. the rate of filtration was 0 in both eyes. At 35 mm. Hg., intra-ocular pressure the filtration from the eserine eye was 11 cc., and from the atropinized eye 8 cc., and this difference augmented as the pressure was raised to 65 mm. Hg. I believe our experiments would be in accordance with clinical

observations as to the influence of these drugs on normal and glaucomatous eyes.

I might finally say a few words on one or two other points which have been raised by Professor Hill. He drew attention to the hæmorrhage into the eyeball, which occurred in the cat after letting off all the intra-ocular fluid, and then raising the blood-pressure by pressure on the abdomen. On the filtration hypothesis the normal difference of pressure between the blood in the ciliary capillaries and the intra-ocular fluid is about 35 to 40 mm. Hg. I do not think there is any difficulty in understanding why a sudden rise of this pressure to something between 100 and 160 mm. Hg., as would occur under the conditions of Hill's experiments, should cause actual rupture of the capillaries and hæmorrhage into the eyeball.

With regard to the question of absorption from the eyeball, he pointed out that the spaces of Fontana in the canal of Schlemm were separated from the anterior chamber by epithelium. The same condition obtains everywhere in the lymphatic system. The lacteals in the villi have a continuous endothelial coat, and the endothelium of the diaphragm is continuous over the stomata, and yet particles of Indian ink or milk globules pass easily by these stomata into the underlying lymphatics. The apparently continuous endothelium does not stop the passage of fluid or even the fine particles. Filtration through it may occur without any rupture of the membrane.

Mr. Priestley Smith said the subject was a large one; he would not enter on the question of the accommodation of the eye, though he would have liked to criticize what had been said, but would confine himself to one or two points connected with the physiology of the intra-ocular pressure. The pressure in the healthy eye varied in different persons, and in the same person at different times. There was no absolute normal. What was normal for one eye might be abnormal for another. The average was about 25 mm. Hg. Healthy eyes doomed to excision by reason of orbital tumor had been tested by the manometer with this result. A pressure of 60 mm. Hg., which Dr. Hill had found under certain circumstances in the eyes of animals, was much above the normal for animal and man. In the human eyes it was equivalent to a severe glaucoma.

Dr. Hill maintained that the intra-ocular pressure was equal to the blood-pressure in the veins and capillaries within the eye. The speaker thought that it was equal to the venous pressure at

certain points only—viz., at those points where the veins left the eye, but that elsewhere, and especially in the capillaries which produce the intra-ocular fluid, the blood-pressure was higher than the intra-ocular pressure, which he would call the chamber pressure. Dr. Hill's observation had shown that the intra-cranial pressure was equal to the blood-pressure in the torcular Herophili, but he had not shown that the blood-pressure in the torcular was equal to that in the veins in other parts of the skull, or in the capillaries; if there was no difference of pressure throughout these vessels there would be no movement of the blood, as Professor Starling had pointed out. In a considerable number of healthy human eyes the behavior of the central vein of the retina close to its point of exit showed a delicate balance between blood-pressure and chamber pressure. With each incoming arterial wave the chamber pressure rose above the blood-pressure at this point and emptied the vein; between the waves it fell below the blood-pressure and the vein refilled; the two were in approximate equilibrium, and each in turn gained the mastery. In the majority of eyes the blood-pressure was a little the higher of the two, and the arterial impulse transmitted by the vitreous was not sufficient to empty the vein even close to its point of exit. Further from the point of exit, and especially in the capillaries of the ciliary body, the blood-pressure must be higher still.

He would like Dr. Hill's opinion as to the probable pressure in the arteries where they enter the eye. [Dr. Hill: You may take it as 100.] Taking the pressure as 100 where the blood enters, and at 25 where it leaves the eye, there is a fall of 75 mm. Hg. Where did this fall take place? It was probably not very sudden. It was important to remember the laws governing the pressure of a fluid moving through a tube. In a horizontal tube of uniform calibre the pressure falls continuously and evenly throughout. In a tube of varying calibre it is modified, and in places the fall may be converted into a rise. Where the tube is larger, the fluid moves more slowly, making less pressure in the forward direction and more against the containing wall; where it is smaller the fluid moves more quickly, the forward pressure is greater, the lateral pressure less. In a vascular area like that of the uveal tract, the aggregate transverse section of the capillaries were greater than that of the arterioles feeding them, and this ensured a slower movement and a greater pressure in the capillaries than would be present if the transverse

sections were equal; the pressure might even be greater than that in the arterioles. Professor Starling had pointed out that the capillary wall was thick enough to support such pressure, and had shown reasons of a different kind for assuming that the capillary pressure must be considerably higher than the chamber pressure—perhaps as high as 60 mm. Hg.—in order to produce the intra-ocular fluid.

It was generally stated that the ciliary processes provided the intra-ocular fluid. He thought it better to say the ciliary *body* so as to include the *pars plana*. The processes were obviously fitted by structure and position to supply the aqueous chamber; nearly the whole of their surface was in open communication with it. The *pars plana* was in intimate relation with the vitreous body. It was thickly studded with the "glands" of Treacher Collins. It was readily reached by staining fluid injected into the vitreous. Morbid exudation could frequently be traced from the *pars plana* into the vitreous. It seemed probable, therefore, that this part of the ciliary body secreted the fluid of the vitreous.

The tension of the eye was maintained by the intra-ocular fluid. The term "intra-ocular tension," often used by writers, was apt to confuse. We ought, he thought, to speak of the tension of the tunics and of the pressure within the eye—or of the ocular tension and of the intra-ocular pressure.

It was sometimes stated that the wall of the eye was practically a rigid case. This was misleading. The tunics were pliable, distensible, and elastic. When a finger was pressed upon the eye the wall was dimpled; the fluid displaced by the dimple was found room for elsewhere. When the finger was removed the dimple disappeared. This proved the distensibility and the elasticity of the tunics. Accurate measurements had been made to ascertain the amount of distension which occurred under a given increase of the intraocular pressure. Errors easily arose through lack of clear perception of such points. For example, it had been contended that increase in the tension of the eye was due to an increase in the pressure of the contents and *not* to an increase in their volume. But the two things are inseparable. An increase in the tension of the eye indicates an increase of the intra-ocular pressure, and it also indicates an increase in the volume of the contents, for neither can occur without the other.